Measurement technique and variation in intramucosal pH

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Summary

We have calculated gastric intramucosal pH (pHᵢ) from Trip catheter saline tonometered samples in two patients undergoing ventilation using four different sampling techniques, each repeated five times. pHᵢ was calculated from measurement of PᵦCO₂ in tonometered saline (TCO₂). TCO₂ was measured immediately, and then at 6-min intervals for 30 min. Variation in measurement was greatest for capped syringes stored at room temperature, and least when stored uncapped on ice. TCO₂ always decreased significantly within 12 min. The mean difference in pHᵢ (all sampling techniques) over 30 min was 0.1005 pH units. The results indicate that the calculated pHᵢ was subject to variation as a result of both the method of sample storage and delay in measurement. An error of ±0.1 pH units may have clinically important implications if pHᵢ is used to monitor either severity of illness or efficiency of resuscitation. (Br. J. Anaesth. 1996; 76: 563–564)

Key words

Gastrointestinal tract, pH. Monitoring, pH oesophageal.

The use of a Trip tonometer (Tonometrics, Inc., Bethesda, MD, USA) to measure gastric intramucosal pH (pHᵢ) is a recent innovation in intensive care medicine. A Trip nasogastric catheter incorporates a saline-filled balloon, which lies in contact with the gastric mucosa. Diffusion of carbon dioxide from gastric mucosa, into saline, allows for its subsequent measurement (TCO₂) using a conventional blood-gas analyser. An arterial blood-gas sample is obtained concurrently, and the measured bicarbonate (HCO₃⁻) used to determine pHᵢ indirectly using a slide rule nomogram, supplied with the tonometer. pHᵢ is said to be an indicator of organ-specific oxygen and substrate delivery and may reveal splanchnic hypoperfusion, a situation of specific oxygen and substrate delivery and may indicate that the calculated pHᵢ is subject to variation as a result of both the method of sample storage and delay in measurement. An error of ±0.1 pH units may have clinically important implications if pHᵢ is used to monitor either severity of illness or efficiency of resuscitation.

Methods and results

Local Ethics Committee approval was obtained before the study. Measurements were made in two stable patients undergoing ventilation who received i.v. H₂ receptor antagonists (ranitidine) throughout the period of investigation. Nasogastric feeding was suspended for 2 h before sampling. The Trip saline prime was allowed to equilibrate in vivo for 90 min before analysis.

Arterial blood-gas and tonometered saline samples were obtained and processed by one investigator using an IL 1312 blood-gas analyser, located within the intensive care unit (ITU). The Trip catheter was re-primed immediately after sample withdrawal.

TCO₂ was measured within 30 s of sampling (0 min), and arterial blood-gas 3 min later, between which the analyser self-calibrated automatically. The measurements were then repeated on the same sample pair, every 6 min for a total of 30 min, during which each individual sample was stored under one of four conditions (all collected in a 2-ml disposable plastic syringe): (1) capped, room temperature; (2) capped, on ice; (3) uncapped, room temperature; and (4) uncapped, on ice.

Accuracy of timing between sampling was ensured by the use of a stop clock and verified by the results print-out from the automatic blood-gas analyser. Each condition was studied exclusively five times during a single 24-h period, producing a total study period of 4 days. Before each series of measurements the blood-gas analyser was subject to a two-point calibration. Under these conditions the reproducibility of recurrent PᵦCO₂ measurement (equivalent to electrode re-calibration drift) was less than 0.01 kPa.

All measurements within each condition were averaged and mean values at time = 0 min and time = 30 min were compared using a paired t test. P < 0.05 was considered significant.

The influence of time on TCO₂, pHᵢ and arterial HCO₃⁻ is shown for each condition in table 1. The mean value of TCO₂ at t = 0 varied by as much as 1 kPa depending on the sampling condition, but the subsequent downward trend over 30 min was seen with each technique, reaching significance at 12 min.
(P < 0.05), pH increased under all conditions, with highly significant and clinically important increases at 30 min (P < 0.02).

The initial calculated pH, of 7.365 (mean of all values) increased by a mean of 0.1005 pH units at 30 min (minimum increase 0.086, uncapped ice; maximum 0.122, capped, room temperature), as a result of changes in measured TCO2. The increases were significant at 12 min in all but one condition (uncapped ice), which became significant at 18 min. There was no statistically significant change in measured arterial bicarbonate over 30 min, with the exception of capped ice samples (P = 0.004).

Comment

We have demonstrated that TCO2 measurement and subsequent pH calculation is subject to variation, depending on the sampling technique. The variation in mean TCO2 at t = 0 is explained, we believe, by our methodology which studied each individual sampling technique on a different day, and was therefore subject to patient variation. However, a subsequent drift in measured TCO2 occurred with all four conditions. This corresponded to a change in calculated hydrogen ion concentration over 30 min of 24.6 %, 21.7 %, 18.5 % and 18.1 % for conditions 1 to 4, respectively (mean of all four sample types = 20.72 %), the change in acidity being disguised by the unit of measurement (pH).

In practice, measurements of arterial blood gases are often delayed. Our results demonstrated that delays in measurement, particularly of TCO2 and hence calculated pH, may have significant implications for the diagnosis and monitoring of resuscitation.

A pH change of 0.1005 units (mean increase in all samples over 30 min) would at a pH value of approximately 7.35, produce alternative values of 7.25 or 7.45. As a pH value of less than 7.35 is considered pathological [4] errors of this nature and magnitude are worrying. Our patients were stable and never clinically shocked and therefore we do not know if the measurement changes that we have documented become more or less significant when pH is pathologically low.

In this study a significant problem with drift in arterial bicarbonate measurement occurred only with the capped ice samples, but as pH is calculated from the HCO3/TCO2 ratio, any drift in this variable is yet another potential source of concern. The accuracy of measurement of TCO2 can be improved by using a phosphate buffered solution [5] but prompt analysis would still be advised. An additional acknowledged difficulty is the error attributable to the choice of blood-gas analyser [6].

We suggest that strict standardization of the conditions of measurement for both arterial blood gases and the tonometered sample is necessary before results gained from Trip analysis may be usefully applied in, and between, different intensive care patient groups.

References


Table 1 Variation in bicarbonate, tonometered carbon dioxide and pH, at t = 0 and t = 30 with sample technique (mean (SD))

<table>
<thead>
<tr>
<th>Sample technique</th>
<th>HCO3 (mmol litre⁻¹)</th>
<th>TCO2 (kPa)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>t = 0</td>
<td>t = 30</td>
<td>P</td>
</tr>
<tr>
<td>Capped, room temperature</td>
<td>27.820</td>
<td>27.360</td>
<td>0.315</td>
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<tr>
<td></td>
<td>(1.201)</td>
<td>(1.861)</td>
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<tr>
<td>Capped, ice</td>
<td>26.660</td>
<td>26.200</td>
<td>0.004</td>
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<tr>
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<td>(1.137)</td>
<td>(1.153)</td>
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<tr>
<td>Uncapped, room temperature</td>
<td>24.300</td>
<td>23.940</td>
<td>0.266</td>
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<td>(0.644)</td>
<td>(0.594)</td>
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<tr>
<td>Uncapped, ice</td>
<td>24.940</td>
<td>24.760</td>
<td>0.195</td>
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<td></td>
<td>(0.270)</td>
<td>(0.358)</td>
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